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United States Department of Agriculture–Agricultural Research Service research on improving host-plant resistance to pests†‡

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Abstract: Host-plant resistance is an efficient, economical and environmentally benign approach used to manage many pests and diseases of agricultural crops. After nearly a century of research, the resources and tools have become more refined, but the basic tasks in breeding for resistance have not changed. Resistance must be identified, incorporated into elite germplasm, and deployed in a form useful to growers. In some instances, biotechnology has expedited this process through incorporating a foreign gene(s) for resistance into elite germplasm. The USDA Agricultural Research Service (ARS) has made significant contributions in the development of germplasm with resistance to insects, nematodes and plant diseases. Because resistant plant varieties are an essential component of sustainable production systems, ARS is committed to developing techniques and germplasm to help meet this goal.
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1 INTRODUCTION

Competition between and interaction among organisms in nature results in natural selection for traits that confer an advantage. As a result of such interactions between plants and their natural enemies, plants have developed defensive mechanisms, both chemical and physical, to minimize pest damage. Since man began to plant and gather food, he has intentionally or inadvertently selected seed from the most vigorous or least damaged plants to propagate his crops. Thus, man was selecting for resistance to insects and plant diseases or for favorable agronomic traits. During the latter part of the 19th century and throughout the 20th century, scientists have exploited natural resistance to improve crop varieties. As a result, breeding for plant resistance to insects/mites and plant pathogens has been one of the major success stories in the control of several key pests of agricultural crops in the USA.

Scientists with the USDA- Agricultural Research Service (ARS) have played a significant role in the identification and development of plant germplasm with resistance to plant pathogens and insects. The National Genetic Resources Program (NGRP) was established in 1990 as a cooperative federal and state effort to preserve genetic diversity. As part of the NGRP, the National Plant Germplasm System has the mission to acquire, store, propagate, evaluate and distribute plant germplasm collected around the world. Whenever a serious pest problem arises or a foreign pest is introduced, an evaluation of germplasm maintained at these repositories to identify resistance to the pest is often the first course of action. Thus, germplasm in the repositories has proved invaluable as a source of genes for resistance to pests of many important agricultural crops grown in the USA.

The ARS has played a key role in plant resistance research since its establishment in 1953, a role that continues into the 21st century. Acknowledging that the agency does not function within a vacuum, the research discussed herein represents collaborative research efforts in which ARS scientists play a vital role. This review is not intended to be all-inclusive, but only to highlight selected ARS plant resistance programs that have made significant contributions in the management of key agricultural pests.

2 OVERVIEW OF INSECT RESISTANCE IN SELECTED AGRICULTURAL CROPS

2.1 Corn earworm

The corn earworm, *Helicoverpa zea* (Boddie), is native to the Americas and occurs wherever corn,

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Zea mays (L), is grown. Adult females prefer to lay eggs on fresh silks and emerging larvae move from the exposed silks to a more protected position in the silk channel formed by the husk extension. If silk quantity is sufficient, larval development may be completed in the silk channel; but, if the amount of silk is limited or if the husks are sufficiently loose, larvae will move to and feed on developing kernels resulting in significant economic impact. In addition to direct kernel damage, corn earworm damage enhances introduction of secondary pests and micro-organisms such as *Aspergillus flavus* (Link) and *Fusarium moniliforme* Sheldon which produce mycotoxins. In the southeastern USA, susceptible corn hybrids serve as a nursery for the development of large corn earworm populations, which not only cause considerable damage to corn, but produce large populations of adults which infest other crops and subsequently cause substantial economic losses in cotton (*Gossypium hirsutum* L), peanut (*Arachis hypogaea* L), sorghum (*Sorghum bicolor* L), soybean [*Glycine max* (L)], and many vegetable and ornamental crops.

Host plant resistance to corn earworm is due to maysin, a C-glycosyl flavone, and related compounds in the silks that inhibit corn earworm larval growth.[1] Upon wounding of silk tissue, such as with insect chewing, maysin and related compounds are believed to be oxidized by polyphenol oxidases to quinones, which are responsible for the silk-browning reaction.[2,3] In the larval gut, quinones apparently bind to -SH and -NH_2 groups of free amino acids and proteins, reducing their availability to the insect and thus inhibiting larval growth and development.[4]

Quantitative trait locus methodology has been used to identify corn chromosome regions associated with silk maysin concentration. In the population (GT114 × GT119) F2, Byrne *et al*[2] studied maysin inheritance by associating phenotypic values of individual plants with genotypic variation at flavonoid pathway loci. Using RFLP markers, they found that the *p1* region of chromosome 1 accounted for 58% of the phenotypic variance for the trait and detected a second QTL on the short arm of chromosome 9 that showed significant epistasis with *p1*.[2,5] Lee *et al*[6] demonstrated that the primary locus controlling the synthesis of apimaysin is located on maize chromosome 5. Guo *et al*[7,8] documented that the interaction between *p1* and *a1* express quantitative genetic control over maysin, apimaysin, methoxymaysin and chlorogenic acid, and confirmed that *p1* and *a1* are major QTLs controlling maysin concentration in populations (GE37 × 565) F_2 and (SCI 02 × B31857) F_2. Other loci with significant associations with resistance in corn to the corn earworm and maysin production include *umc105a* on chromosome 9S,[2] *asg20* on 2L,[9] *wx1* located on 9S,[6,9] *bnl5.71* on 5C-5L,[6] *umc85* on 6S,[6] *npi286* on 1S,[8] and *csu1066* on 2C-2L.[10,11]

2.2 Fall armyworm

Corn and grain sorghum are grown on more than 95 million acres in the USA. The fall armyworm, *Spodoptera frugiperda* (JE Smith) is one of the most economically damaging insect species of the tropical and subtropical regions of the Western Hemisphere and is an especially important pest of corn and sorghum. Average annual crop losses to the fall armyworm in the USA exceed $300 million, but during particularly severe outbreaks losses attributed to this pest may exceed $500 million annually.

ARS scientists Scott, Davis and Williams released the first corn germplasm with resistance to fall armyworm.[12–14] Since then, numerous inbreds have been developed from Antigua germplasm. Factors associated with the resistance, such as high hemicellulose content, low protein content and leaf toughness, are correlated with reduced larval growth.[15] Recently, a gene coding the 33-kD cysteine proteinase has been cloned from corn genotypes resistant to the fall armyworm.[16] When larvae were reared on callus expressing the proteinase, their growth was inhibited 60 to 80%.[17]

2.3 European corn borer

The European corn borer, *Ostrinia nubilalis* (Hübner), was introduced into the USA in the early 1900s and has spread across the corn-producing areas of the country from the east coast to the Rocky Mountains. Estimated annual losses in the US Corn Belt alone range from $200 to $500 million annually.[18] Losses result from physiological damage due to feeding injury to the plant, reduced quality due to direct kernel damage and to dropped ears.

Extensive host plant resistance research on the European corn borer identified antibiosis resistance[19] due to high concentrations of 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA) in the leaves of mid-whorl stage plants.[20] Commercial seed companies have incorporated this resistance into commercial corn varieties.[18] In addition, European corn borer resistant composite populations Mo-2ECB and Mo-2 ECB-2, and inbreds Mo45, Mo46 and Mo47 have been released with excellent resistance to both leaf feeding and sheath and collar feeding.[18]

More recently, resistance to the European corn borer was identified in Peruvian corn germplasm[21] and the GEM (Germplasm Enhancement of Maize) corn collection.[22] Antibiosis and feeding non-preference were identified as the mechanism of resistance in the Peruvian germplasm.[23] All of the Peruvian lines had low concentrations of DIMBOA in the leaves, indicating that another mechanism was responsible for the resistance. Several of these corn lines also had resistance to other pests.[24] A backcross breeding program with this germplasm using B94 or B97 as the recurrent parent was extremely successful, with 15 lines identified with leaf feeding resistance and eleven lines with both leaf feeding and sheath/collar feeding resistance to the European corn borer.[21]

Two of these lines [(PI 503720 × B97)//B97 and (PI 503806 × B94)//B94] were also resistant to leaf feeding by the fall armyworm and another [(PI 503731 × B94)//B94] was resistant to silk feeding by the corn earworm.[25] Thus, several of these lines offer the potential to develop commercial lines of corn with multiple pest resistance.

2.4 Hessian fly

The Hessian fly, *Mayetiola destructor* Say, a serious pest of small grains worldwide, was introduced into the USA in approximately 1776. It has since spread throughout most of the USA and Canada and is one of the most destructive pests of small grain.[26] Excellent reviews on the Hessian fly were published by Ratcliffe and Hatchett[26] and Ratcliffe *et al*[27] Most of the following was taken from these reviews.

Feeding injury by Hessian fly larvae on wheat in the late fall–early winter results in stunted plants which appear more erect, shorter and have darker green leaves than uninfested plants. Feeding injury by larvae of the spring generation interferes with elongation of nodes and transport of nutrients, resulting in reduced yield, poor quality grain and broken culms which increase harvest losses.

A combination of different methods is used to manage Hessian fly populations and damage, including delayed seeding of winter wheat to avoid peak emergence of adults, destroying volunteer wheat and planting resistant varieties, which remains the primary method for avoiding economic loss to Hessian fly. As noted by Ratcliffe *et al*,[27] 'Approximately 70 years of research has led to the identification and phenotypic characterization of 29 resistance genes from common and durum wheats, wild wheat relatives and rye, *Secale cereale* L'. The primary mechanism of resistance to Hessian fly is antibiosis which results in death of first-instar larvae, but non-preference has also been documented for genotypes with greater leaf pubescence which reduces oviposition by females. Ratcliffe and Hatchett[26] list the source of resistance, chromosome location, and selected reference for 27 of the 29 genes for Hessian fly resistance. Resistance to the Hessian fly in wheat is dominant to partially dominant and conditioned primarily by single genes, although duplicate and multiple genetic factors have been identified.

Since resistance to Hessian fly in most wheat cultivars is conditioned by a single gene, virulent populations of this insect have readily developed. Currently, 16 Hessian fly biotypes, identified on the basis of virulence or avirulence of larvae to four wheat differentials carrying different resistance genes, have been identified. Genetic studies have shown that virulence in the insect is conditioned by a single recessive autosomal or sex-linked gene, and that the genetic interaction between resistance in the plant and virulence in the insect exhibits a gene-for-gene relationship. Therefore, because of the recessive nature of the insect virulence to the resistant plant, the virulence can only be expressed when the insect is homozygous for this trait. Furthermore, because numerous plant genes for resistance have been identified, Hessian fly biotypes have been managed by a sequential release of cultivars with different genes for resistance to the insect. RAPD markers linked to plant genes for Hessian fly resistance have been identified[28,29] that expedite marker-assisted breeding to incorporate multiple resistance genes into a single genotype. New resistance management strategies to slow development of virulent biotypes are being developed.

2.5 Greenbug and Russian wheat aphid

Several species of aphids are important, primary pests of small grain grown in the USA. The greenbug, *Schizaphis graminum* (Rondani) feeds on sorghum, wheat, barley and other small grains and was the most important small grain aphid pest until the discovery of the Russian wheat aphid, *Diuraphis noxia* (Mordvilko), in Texas in 1986.[30] Since its introduction, the Russian wheat aphid has spread throughout the Western USA and has become a serious, perennial pest of both wheat and barley.[30,31] The small grains germplasm collection maintained by USDA-ARS has proved to be of vital importance in the identification of resistance to both important aphid pests.

At the time of its introduction, all commercial cultivars of wheat and barley were highly susceptible to the Russian wheat aphid.[30] Subsequently, the entire working collection of wheat and barley from the USDA-ARS National Small Grains Collection was screened for resistance, and several different sources of resistance were identified. Initial screening efforts identified resistance in five barley accessions from Afghanistan (PIs 366444, 366447, 366449, 366450 and 366453) and one accession from Iran (CI1412). PI 366450 had a high level of resistance and was selected for use in an accelerated breeding program to provide resistance to this insect in barley. Selection for uniformity of resistance led to the release of STARS-9301B.[32] Resistance is due to tolerance, antibiosis and antixenosis, resulting from the action of two genes, *Dnb1* and *Dnb2*,[33] controlled by recessive epistasis of the dominant gene *Dnb2* on the incompletely dominant *Dnb1*. Another source of resistance, STARS-9577B, was released in 1999.[34] Resistance in this line is primarily due to tolerance, but a low level of antibiosis is also present.

Several important sources of resistance to the Russian wheat aphid have been identified in wheat. Resistance in PI 140207 and PI 137739 is due to antibiosis and is controlled by a single dominant gene, *Dn1*.[30] Porter and Webster[31] reported that a 24-kD protein complex was inhibited in a susceptible wheat genotype after the Russian wheat aphid fed, but persisted after feeding in PI 140207. They concluded that feeding by the Russian wheat aphid selectively inhibits normal metabolic functions in susceptible plants, but not in the resistant genotype. Webster and

Porter[35] found that resistance to the Russian wheat aphid in STARS-9302W does not confer resistance to other important species of aphid, the greenbug, bird cherry-oat aphid, *Rhopalosiphum padi* (L) or the yellow sugarcane aphid, *Sipha flava* (Forbes).

The greenbug is still a serious pest, especially in the Southern Great Plains where periodic outbreaks result in millions of dollars in losses due to damage and costs of control.[36,37] It has been able to adapt to changes in the environment and resistance in hosts, resulting in the development of several biotypes. Currently 11 biotypes of the greenbug have been identified.[38] Six sources of resistant wheat, each governed by a different, single gene, have been identified: DS 28A,[38] PI 578213 (Amigo),[39] PI 268210 (Largo, CI 17895),[37] CI 7959,[40] CI 17882[40] and PI 561948 (GRS 1201).[37] Porter *et al*[38] chronicle the development of the different greenbug biotypes and the reaction of each biotype to the six sources of resistance in wheat. Currently, there are three predominant greenbug biotypes, E, I and K. Webster and Porter[37] reported that 'GRS 1201' and 'Largo' were resistant to these three biotypes, but 'GRS 1201' had a much higher level of combined resistance than did 'Largo.' Pyramiding genes in wheat for greenbug resistance did not enhance resistance to the various greenbug biotypes over that provided by a single gene for resistance.[41]

Dubcovsky *et al*[42] reported the translocation of a greenbug resistant gene *Gb5* from *Triticum speltoides* (Taush) Gren to wheat. The translocated *Gb5* gene was located on the long arm of chromosome 7A, and RFLP markers were identified to assist in efficient marker-assisted breeding to transfer the resistance gene to new cultivars with resistance to the greenbug.

2.6 Insect resistance in transgenic crops

Transgenic crops expressing a protein from the bacterium *Bacillus thuringiensis* Berliner *(Bt)* have been commercially available since the mid-1990s and have been readily accepted by both the American producer and consumer. The *Bt* bacterium is ubiquitous and is unique in that it produces a crystalline (cry) protein during sporulation that is toxic to certain insects. In 2001, genetically engineered crops were grown on 52.6 million hectares (130 million acres) worldwide, up 19%, or almost 20 million acres from 2000.[43] Of this total, 88.2 million acres were planted to transgenic crops in the USA in 2001 and included soybean, cotton, corn and potato (*Solanum tuberosum* L). Of the total acreage planted to transgenic crops, herbicide resistance accounted for 77%, *Bt* crops for 15%, and stacked genes for herbicide and insect resistance accounted for 8%. Growers who planted *Bt* cotton reduced insecticide applications by an estimated 2.7 million pounds and made 15 million fewer insecticide applications each year than those that planted conventional cotton.[44] The US Environmental Protection Agency (EPA) recently renewed registration of *Bt* crop varieties for another 7 years.[45]

ARS scientists have played an important role in the evaluation and development of genetically engineered crops, development of insect resistant management (IRM) programs, monitoring for resistance to the cry proteins and monitoring for adverse effects on non-target organisms and the environment. BollGard cotton containing a *cry1Ac* gene was the first transgenic crop commercialized. It is very effective in controlling the tobacco budworm, *Heliothis virescens* (Fab), but is less effective in controlling the cotton bollworm, *H zea*.[46] Recent evaluation of BollGard II transgenic cotton containing both Cry1Ac and Cry2Ab proteins showed control of cotton bollworm, fall armyworm and beet armyworm, *S exigua* (Hübner), better than control of these insects with BollGard which only expressed Cry1Ac.[47,48] Scientists in the NC205 regional research committee (Ecology and Management of European Corn Borer and Other Stalk-Boring Lepidoptera), which included ARS scientists, led the effort to establish a practical IRM program for corn growers. A unified approach to *Bt* corn IRM has gained wide stakeholder acceptance and increased grower compliance. Close collaboration with EPA has allowed the NC205 committee to identify important research areas which the EPA has addressed in its amended registration document for *Bt* corn.[45]

In both laboratory and field tests, transgenic field corn was almost immune to damage by the southwestern corn borer, *Diatraea grandiosella* Dyar, and highly resistant to the corn earworm and fall armyworm.[49–51] A combination of traditional resistance plus *Bt* transgenes was more effective in control of the fall armyworm than either component alone.[51] Transgenic sweet corn containing a gene for Cry1Ab production was extremely resistant to the corn earworm and highly resistant to the fall armyworm.[52,53] Resistance to the *D grandiosella* in *Bt* hybrid field corn did not reduce aflatoxin contamination when plants were inoculated with *A flavus* spores or *A flavus* spores and *D grandiosella*.[54] However, reduced *Fusarium* ear infection and fumonisin in the kernels was noted in *Bt* corn lines expressing Cry1Ab protein as compared with near-isogenic, non-transformed corn lines.[55–57]

Recent research showed that current *Bt* proteins produced in EPA-approved commercial corn hybrids pose a minimal threat to Monarch butterfly, *Danaus plexippus* (L), larvae. Results from collaborative ARS and university studies suggest that *Bt* pollen densities in excess of 1000 grains cm^{-2} would be required to have an adverse effect on Monarch larvae.[58] Under field conditions, pollen contamination of milkweed average 10 to 425 grains cm^{-2}.[59] Thus, the 2-year collaborative research project suggested that *Bt* corn pollen produced by current EPA-approved commercial hybrids would have a negligible effect on Monarch populations.[60]

3 OVERVIEW OF RESISTANCE TO PLANT-PARASITIC NEMATODES

Plant-parasitic nematodes cause an estimated yield loss of $8 billion annually in the USA.[61] Crop rotation, nematicides and host-plant resistance are the primary management tactics which growers use to reduce yield losses. Of the three tactics, host-plant resistance is preferred because it suppresses nematode reproduction, shortens the length of rotation, reduces the need for costly nematicide, does not require specialized equipment, and keeps seed costs similar to that of susceptible cultivars.[62] Current research programs within ARS are seeking to improve nematode resistance in cotton,[63] peach rootstocks [*Prunus persica* (L) Batsch],[64] peanut,[65] pearl millet [*Pennisetum glaucum* (L) R Br],[66] potato,[67] soybean,[68,69] sugar beet [*Beta vulgaris* (L)],[70] tall fescue (*Festuca arundinacea* Schreb)[71] and vegetables [carrot (*Daucus carota* L), bell pepper *(Capsicum annuum* L) and hot pepper].[72–74]

Nematologists within the ARS have worked closely with plant breeders (ARS and university) to identify and release resistant germplasm and cultivars. Resistant genotypes are typically identified based on nematode reproduction relative to a known susceptible genotype. Plants that support low nematode reproduction are considered highly resistant, while plants that support intermediate reproduction are considered moderately or partially resistant. Screening plants for resistance is both time and space consuming. In greenhouse studies, reproduction is generally measured 1 to 3 months after nematode inoculations. Several replicates of each plant genotype are necessary, particularly with moderately-resistant genotypes, because nematode reproduction is highly variable even in the same plant background.

In the last decade, several ARS programs have identified molecular markers for nematode resistance genes. Closely linked markers can be used in breeding programs to quickly identify resistant genotypes (marker-assisted selection). Molecular markers for the H_1 gene in potato are currently being used to screen segregating populations for resistance to the potato cyst nematodes, *Globodera rostochiensis* (Wollenweber) Behrens and *G pallida* (Stone) Behrens, in a cooperative USDA/Cornell breeding program.[75] The ability to pre-screen a large number of potato genotypes for resistance in the absence of *Globodera* spp, at many locations, is especially important because the 'golden nematode', *G rostochiensis*, is a quarantine pest with a limited distribution in North America. Molecular markers close to two loci conferring resistance to the soybean cyst nematode (*Heterodera glycines* Ichinohe) have also been identified in soybean.[76,77] These two loci designated Rhg_1 and Rhg_4 are responsible for most of the resistance to the soybean cyst nematode, which is the most damaging pest of soybean in the USA and worldwide. Recently, molecular markers have been identified for the *Mj-1* locus[78] conferring resistance to the root-knot nematode, *Meloidogyne javanica* (Treub) Chitwood, in carrot. Root-knot nematodes are devastating pests of carrot because even low populations of these nematodes can result in reduced marketability from galling and forking of the tap root.

A number of ARS programs have recently characterized the genetics, expression and breadth of nematode resistance. These studies increase our understanding of the resistance and will ultimately improve utilization of resistant cultivars and resistance genes. Recent advances in characterizing nematode resistance in pepper, peach and soybean are highlighted below.

3.1 Pepper

Root-knot nematodes (*Meloidogyne* spp) are serious pests of pepper. Several hot pepper (*C chinense* Jacq) genotypes and two bell pepper cultivars, 'Charleston Bell' and 'Carolina Wonder', with resistance to *Meloidogyne incognita* (Kofoid & White) Chitwood, were also found to be resistant to *M arenaria* (Neal) Chitwood race 1 and 2, and *M javanica*, but susceptible to *M hapla* Chitwood.[72,74] The resistance is conferred by a single dominant gene designated *N* in bell pepper and by a single dominant gene, allelic to the *N* gene in hot pepper.[79,80] At high temperatures (32 °C), the resistance in both bell and hot pepper was somewhat compromised, but even at high temperatures nematode reproduction on the resistant plants was only 20% that of the susceptible plants.[81,82] Nematode-resistant pepper cultivars should provide a viable alternative to preplant fumigation with methyl bromide.

3.2 Peach

'Guardian' is a peach rootstock with tolerance to peach-tree-short-life (PTSL), a disease complex affecting trees throughout the southeastern USA. However, root-knot nematodes *(M incognita* and *M javanica*) are also a problem in this region, causing tree stunting, loss of vigor, and early defoliation of peach trees. Recently, 'Guardian' was shown to be resistant to a population of *M incognita* from Georgia and to *M javanica* from North Carolina, but only moderately resistant to a population of *M javanica* from California and susceptible to *Meloidogyne* sp from Florida.[64,83] The lesion nematode, *Pratylenchus vulnus* Allen & Jensen, is also found in peach orchards; however, little is known about its economic importance in the southeastern USA. In a study to determine the relative susceptibility of 'Guardian', 'Lovell' and 'Nemaguard' to the lesion nematode, the rootstocks were found to support nematode reproduction; moreover, the nematode had a deleterious affect on tree growth.[84] Although peach rootstocks with resistance to PTSL and root-knot nematodes are available, it is clear from the above studies that additional sources of nematode resistance are needed.

3.3 Soybean

Several genes are involved in resistance to the soybean cyst nematode, *H glycines: rhg1, rhg2, rhg3* and *Rhg4.*

Using microarrays to monitor gene expression, the number of genes induced by twofold in the presence of *H glycines* were greater in resistant 'Peking' than in susceptible 'Kent'.[85] Some of the genes induced specifically in the resistant soybean are defense-related genes, possible regulatory and transcription factors, and several are unknown genes. Efforts are underway to clone, using map-position-based techniques, the *Rhg4* locus on linkage group A2 which confers resistance to race 3 of the soybean cyst nematode.[86] Some markers contained in the *Rhg4* region of the resistant PI 437654 were separated by a greater physical distance than the same region in the susceptible 'Williams 82'. In addition, PCR primers amplified bands in PI 437654 that were not amplified in 'Williams 82', suggesting that there is an insertion in the PI which may condition resistance to *H glycines*.[86]

4 OVERVIEW OF RESISTANCE TO PLANT DISEASES

There is a considerable effort within ARS aimed at improving resistance to diseases. Highlighting significant progress in disease resistance is a broad assignment. The arena encompasses resistance to diverse parasites (fungi, viruses, bacteria, phytoplasmas, parasitic weeds and nematodes, of which the latter was discussed earlier) of diverse hosts (herbaceous annual and herbaceous and woody perennials species used for grain, forage, vegetables, timber, fruit, nuts or ornamental purposes). Research into disease resistance is expanding across the spectrum of perhaps hundreds of pathosystems at all levels. Several criteria can be used to define significant research within these pathosystems. For commodity crops produced in surplus, a greater understanding of resistance to pests can be significant if it uncovers fundamental biological mechanisms of resistance. Perhaps equally significant is research which represents progress in developing resistance to diseases which have historically required pesticides for their control, or for which few or no sources of genetic resistance have been identified previously. Based on these criteria, the research presented here illustrate the principal processes in and obstacles to use of resistance for disease control.

Identifying new sources of resistance remains an important priority for many pathosystems, and can be subject to various complications. For many diseases, highly effective resistance is not known, so quantitatively expressed resistance is being sought. Potatoes with verticillium wilt resistance are not commercially available; however, clones with resistance to *Verticillium dahliae* Kleb and *V albo-atrum* Reinke & Berthold have recently been identified and the commercial value of resistance was demonstrated.[87] In some instances, pathogens are transmitted by a vector, and identifying resistance to the pathogen can be confounded by the reaction of the host to the vector. Resistance to corky ringspot disease (caused by tobacco rattle virus) has recently been identified in potato germplasm. The germplasm was shown to be resistant to the virus, rather than to its vector, the stubby root nematode [*Paratrichodorus allius* (Jensen) Siddiqui].[88] Some plant diseases are complexes, resulting from the interaction of two pathogens. Cowpea stunt results from the synergistic coinfection of both cucumber mosaic virus (CMV) and blackeye cowpea mosaic virus (BICMV). Resistance to CMV has never been identified until recently,[89] and this germplasm will be valuable for breeding new stunt-resistant cowpea varieties.

Where resistance does not exist within adapted or exotic germplasm, accessions of wild and related species are then explored as sources of resistance. Use of this germplasm may be problematic due to genomic differences, endosperm degeneration in hybrids or meiotic irregularities and linkage drag associated with translocations. In spite of these difficulties, these genetic resources are invaluable if resistance has not been identified in cultivated species. Recent advances include identification of resistance to Sclerotinia stem rot [*Sclerotinia sclerotiorum* (Lib) de Bary] of soybean in perennial *Glycine* species,[90] resistance to bacterial angular leaf spot (*Xanthomonas fragariae* Kennedy & King) in *Fragaria virginiana* Duchesne, a wild progenitor species of cultivated strawberry (*Fragaria* × *ananasa* Duchesne),[91] and resistance to the parasitic weed *Striga hermonthica* (Del) Benth in wild *Pennisetums* which can be used to breed resistant pearl millet.[92]

Where genes for resistance are not known within the crop or among its related species, they may be derived from entirely unrelated species. Transgenic wheat has been developed with a gene encoding a bacterial ribonuclease III. Plants that express this bacterial gene have a reduced accumulation of barley stripe mosaic virions and are asymptomatic after inoculation.[93] An increasingly common approach to breed for virus resistance is to use genes from the pathogen itself. As a recent example of a new resistance, transfer of the gene for the coat protein of the plum pox potyvirus into European plum effectively conferred resistance to the plum pox virus.[94]

Regardless of their origin, genes for resistance to diseases with high epidemic potential must be managed in a manner that preserves their effectiveness. Long-term protection in these systems has depended on identifying and incorporating new resistance genes or combinations into cultivars, which are summarily rendered susceptible by changes in the pathogen population. Alternative strategies are needed to slow or halt the erosion of useful genetic resistance. A new gene deployment strategy has been proposed in the dynamic multi-line population concept,[95] which integrates the best attributes of the gene stacking and multi-line approaches to breeding. Early research results demonstrate its effectiveness and applicability to certain pathosystems.

Efficient selection for resistance during the breeding process is increasingly being facilitated by linkages to markers. When resistance is highly effective, markers

can be useful to combine genes with similar resistance phenotypes. The marker-assisted selection techniques that were refined in systems with easily identified resistance are increasingly being applied to selecting resistances which have a low heritability or whose inheritance has a complex genetic basis. Several advances are being made in identifying markers for resistances with low heritability, including resistance to grain mold in sorghum,[96] Fusarium head blight (*Fusarium graminearum* Schwabe) in wheat,[97] white mold (*Sclerotinia sclerotiorum* (Lib) de Bary) in bean, *Phaseolus* spp,[98] stem rot (*S oryzae* Cattaneo) in rice (*Oryza sativa* L),[99] and brown stem rot [*Phialophora gregata* (Allington & DW Chamberlain) W Gams] in soybean.[100,101] While marker-assisted selection is generally used to select dominant or additive resistance in functionally diploid species, it also shows promise to select resistance in genetically complex systems. Inheritance in alfalfa (*Medicago sativa* L) is complicated by its autotetraploid genome, but markers for resistance to downy mildew (*Peronospora trifoliorum* de Bary) have been identified.[102] The technique is also being applied to marking genes with an epistatic effect on resistance to bean common mosaic potyvirus.[103]

As more markers are identified and the information is consolidated, the potential to assemble any combination of resistance genes is improved. The identification of markers linked to many loci for resistance to many different diseases in common bean[104,105] will accelerate the selection of desirable gene combinations regardless of their source, individual gene action and interactions, and the level of heritability.

Following the identification of loci associated with resistance, sequencing has revealed that the basic structure of some resistance genes have regions that are conserved across different species. Primers have been designed based on those sequences that encode proteins with the conserved nucleotide binding sites and leucine-rich repeats (NBS-LRR class of resistance gene). When these resistance gene analogs are used as probes, locations of related sequences can be identified throughout plant genomes. As the structures within these sites are examined, they are often found to be represented by functional disease-resistance loci embedded within a cluster of non-functional paralogs.[106] It is thought that these gene clusters generate new resistance phenotypes through unequal crossing over, and are maintained because of infrequent recombination. For example, genetic variation within the Mla locus in barley encodes at least 28 different resistance specificities to the powdery mildew pathogen, *Blumeria graminis* (DC) EO Speer f sp *hordei* Em Marchal. This locus is comprised of three distinct families of resistance gene homologues. Compared to the markers adjacent to the Mla resistance gene cluster, recombination is reduced within this region.[107] Proteins encoded by these regions are highly related and can have similar resistance phenotypes, but the alleles can have distinct downstream signaling components which trigger resistance.[108] Loci for genes affecting resistance expression can be located either within resistance gene clusters[109] or at independent loci.[110] Understanding the underlying structure and processes triggering resistance will be useful for manipulating resistance genes for broader effectiveness and greater stability of resistance.

5 SUMMARY OF HOST PLANT RESISTANCE RESEARCH IN ARS

Host plant resistance is an efficient, economical and environmentally benign approach used to manage many pests and diseases of agricultural crops. ARS scientists and their collaborators have made important contributions in the development of resistant germplasm. Germplasm maintained by the National Plant Germplasm System helps assure that genetic diversity is preserved and available for scientific use. Genetic resources are vital to the development of plants with resistance to pests. In times of critical needs, this germplasm is one of the primary sources of genes for resistance. With the inherent genetic variability that exists within crop species, significant advances have been and will continue to be made through traditional breeding efforts. After nearly a century of research the resources and tools have become more refined, but the basic tasks in breeding for resistance have not changed. Resistance must be identified, incorporated into elite germplasm and deployed in a form useful to growers. Improved techniques for identifying, locating and reproducing genes and transforming plants, coupled with a better understanding of the underlying biochemical processes resulting in pest resistance will allow more effective pest control and more sustainable agricultural production systems. ARS has and will continue to make significant contributions toward this goal.

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